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An Epidemiological Analysis

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EXECUTIVE SUMMARY

AMBIENT OZONE AND HUMAN HEALTH: AN EPIDEMIOLOGICAL ANALYSIS

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EXECUTIVE SUMMARY

In June of 1982 Resources for the Future (RFF) began a project for the Economic Analysis Branch of the Environmental Protection Agency's Office of Air Quality Planning and Standards (OAQPS). The purpose of that project was to provide information to OAQPS on the human health benefits that might be associated with possible alternative National Ambient Air Quality Standards for ozone, one of six so-called "criteria" air pollutants. In September of 1983, RFF completed work on a two-volume Draft Final Report on that project, "Ambient Ozone and Human Health: An Epidemiological Analysis." Since that time RFF has conducted additional analysis in response to comments, criticisms, and suggestions arising from the draft report. Many of these comments were offered at a Public Peer Review Meeting held in Raleigh, N.C. on April 3, 1984. This Executive Summary briefly reviews the methodology and conclusions in Volumes I and II, the original draft report, as well as those in the recently completed Volume III, the sequel to RFF's initial work.

Volume I

Volume I of the 1983 Draft Final Report presents the data, methodology and findings of RFF's original epidemiological analysis. Chapter 2 discusses in great detail the health and socioeconomic data used in the study, the air pollution measurements used to link exposure to health status, and the meteorological and other data that were also used. Chapter 3 discusses the methodology used to explore possible links between ozone

and other pollutants on the one hand and acute and chronic illness on the other. Neither chapter is reviewed in detail in this Executive Summary. Rather, we prefer to review here the findings of the "Results" chapter, Chapter 4. However, a brief review of chapters 2 and 3 may prove useful.

The health and socioeconomic data upon which the analysis in Volume I is based come from the 1979 Health Interview Survey (HIS) of more than 110,000 adults and children drawn randomly from the United States (Volume I, pp. 2-1 through 2-21). For each individual included in the HIS, information is elicited on personal characteristics (age, race, sex, etc.), occupational or educational status, and on any of several types of acute health episodes during the two weeks immediately prior to the week of the interview. Also included is information on the presence or absence, as well as the severity, of any chronic illnesses from which the respondents may suffer. For all acute and chronic illnesses, coding in the HIS is according to the standard International Classification of Diseases. Thus, one can identify from the HIS, for instance, the number of "bed disability" days a respondent suffered on account of respiratory disease during a two-week period in 1979. Similarly, one can identify all individuals suffering from asthma, emphysema or other chronic respiratory disease. Finally, the 1979 HIS contained two very useful supplements. The first was a detailed lifetime smoking history for 26,000 of the 78,000 adults interviewed in the HIS. The second was a residential history, again administered to one-third of the adult respondents.

The air pollution data used in the RFF Draft Final Report, and in the more recent Volume III analyses, all come from EPA's SAROAD system (Volume

I, pp. 2-22 through 2-46). Hourly data were used to measure ozone, carbon monoxide, and nitrogen dioxide concentrations. Total suspended particulates and sulfates were measured on a 24-hour basis with the readings being taken every six days generally. Thus, exposure to the latter two pollutants may be measured less accurately than for the former. Before matching these data to individuals (through a process described on p. 2-46), all data were subjected to a variety of tests to eliminate potentially incorrect values or outliers resulting from coding errors. Also described in Chapter 2 are the procedures used to average some of the air pollution data over the two-week recall period used in the acute epidemiological analysis as well as over a six-year period used to facilitate RFF's chronic epidemiological analysis. The data on temperature, humidity, precipitation, windspeed and other weather conditions come from NOAA's meteorological monitoring stations around the U.S.

Finally, Chapter 2 discusses the sources of data on pollen concentrations, the use of gas stoves in kitchen cooking, the average amount of annual paid sick leave permitted workers, and the availability of medical care by geographic area in the United States.

The measures of health status used in the Volume I analyses are, for acute illness, the number of restricted activity days, work loss (or school loss) days, and bed disability days during each individual's two-week recall period. The category "restricted activity day" is an inclusive one that encompasses both work (or school) loss and bed disability days as well as days on which respondents had to curtail their normal activities to some

extent short of missing work (or school) or bed confinement. These latter impairments we referred to in Volume I as "minor restricted activity days." Each type of acute illness was analyzed separately. Also separate analyses were conducted using acute episodes due to all causes, and then episodes limited to respiratory illness alone. In addition, logistic regressions were performed in which the dependent variable indicated whether or not the respondent had at least one day of illness during the two-week period. Logistic regression was used exclusively in the analysis of respiratory and other types of chronic illness. In all cases, children and adolescents (those < seventeen years of age) were analyzed separately from adults.

Chapter 4 of Volume I, wherein are reported RFF's empirical findings, represents the heart of the Draft Final Report. The major part of that chapter is devoted to the analysis of acute illness, particularly respiratory disease, and its possible link to short-term exposure to ozone and other air pollutants. (Throughout the analysis of acute illness, exposure to ozone is characterized by the average over the fourteen-day recall period of the daily maximum one-hour reading at the monitor closest to the respondent's residence; no individuals are included in the estimate sample if they live more than twenty miles from the nearest air pollution monitor.) More attention is devoted to dose-response estimation among adults than among children.

This emphasis on acute illness in adults as related to ozone follows from a strategy adopted throughout Volume I: use sensitivity analysis primarily to test the robustness of any positive and significant

associations discovered between ozone and illness. Since such associations arose almost exclusively among adults and when examining acute illness, the report reflects this emphasis. This approach reflects the fact that both time and resources were finite in the original analysis, as they are in all such undertakings.

Generally speaking, and this is an important qualification, the analysis of acute illness among adults supports the following conclusion: there appears to be no consistent association between average daily maximum ozone concentrations and either work loss days or bed disability days, the two more severe types of acute impairments analyzed in Volume I; however, it was frequently the case that ozone was positively and significantly associated with "minor" restrictions in activity, i.e., those that did not necessitate work loss or bed confinement. While there were few or no exceptions to the former conclusion, there were a number of exceptions to the latter. Nevertheless, when the analysis concentrated on respiratory disease in particular--where air-pollution health effects are most likely to occur--ozone was regularly found to be related in a positive and significant way to either the number of restricted activity days or the likelihood of having at least one during the two-week recall period (see Volume I, Tables 4-7, 4-8 and 4-10). For no other air pollutants was such a regular association found.

Among the sensitivity analyses conducted to test this conclusion were the following: (i) varying combinations of air pollutants other than ozone were included in the estimating equation; (ii) the square of ozone was introduced to examine possible non-linearities; (iii) logistic regressions

were run in place of ordinary least squares; (iv) tighter requirements were imposed on the availability of ozone data (a greater percentage of the maximum number of hourly readings were required); (v) the analysis was restricted to respondents interviewed during summer months; (vi) pollen concentrations were introduced as an additional explanatory variable; (vii) the analysis was restricted to those suffering from chronic illness; (viii) the twenty-mile distance cut-off was tightened to ten and then five miles; (ix) lagged values for ozone (from a previous two-week period) were used to proxy exposure; (x) possible synergistic effects between ozone and other air pollutants were examined; and (xi) separate regressions were run for a set of "self-respondents," i.e., those who answered the HIS for themselves. As suggested above, in some of these sensitivity analyses the ozone variable was not positively and significantly associated with acute respiratory disease (or disease due to all causes). More often than not, however, it was.

The findings in the Draft Final Report regarding acute illness among children can be summarized more readily. Virtually no positive and significant associations were found between ozone and any of the types of acute impairments that were considered. This was the case when the focus was on all types of illness as well as when the analysis focused exclusively on acute respiratory illness. Ozone was associated with school loss at the 10 percent confidence level in one or two models, it should be added, but never at the 5 percent level used as the "cutoff" in Volume I of the report. The only association significant at the 5 percent level occurred in one model exploring ozone and bed disability days due to

respiratory disease. Of the other air pollutants included in the analysis, nitrogen dioxide was found to be positively and significantly related to school loss days among children in the analysis of illness due to all causes. While its coefficient was positive in the analysis of minor restrictions in activity and bed disability, nitrogen dioxide was not significantly associated with these forms of acute illness. The one caveat that should be issued here is this: very few sensitivity analyses were conducted using the children's data set because of the absence of initial findings concerning ozone pollution/acute illness associations. More careful analysis of possible air pollution health effects among children should be conducted.

Volume I of the Draft Final Report also devotes considerable attention to the possible role of ozone and other air pollutants in chronic illness. In this analysis, which begins on p. 4-50, the focus shifts away from the two-week recall period and the number of days of restricted activity experienced by each respondent during that period. Instead, the dependent variable in the chronic analysis is whether or not the respondent has chronic respiratory (or, in some cases, cardiovascular or other) disease. Accordingly, the measurement of ozone and the other air pollutants is no longer specific to the two-week recall period, as it was in the analysis of acute illness. Rather, ozone and other air pollution concentrations are averaged over longer periods of time.

Generally, the longer averaging time is one year. However, a multi-year air pollution data set was created for this project. Thus, in some of the models fit in Chapter 4, individuals are matched to air

pollution concentrations over the six year period 1974-79. The logic behind this is straightforward. Chronic illness, if related to air pollution at all, is likely to result from prolonged exposure over many years. Frequently, a single year's data are used to proxy exposure over a much longer period of time. However, where a longer-term air pollution data set could be assembled, as it was in this study, it is preferable to the use of one year's data. As on the analysis of acute illness, the air pollution data matched to an individual came from the monitor nearest his home, so long as minimum requirements regarding data completeness were satisfied.

In general, the analysis of chronic respiratory and other diseases in adults revealed no pattern of positive and statistically significant associations between illness and ozone or most of the other air pollutants. There were occasional exceptions, some of which are discussed below, but the results contrast with the findings about ozone and minor restrictions in activity due to respiratory disease.

In the initial analysis (see Table 4-13 in Volume I), all pollutants were measured using a single year's data, 1979, to proxy long-term exposure. No significant associations were observed for any of the air pollutants. When the multi-year data set was brought into play, the results changed somewhat (see Table 4-14). In some models, the coefficient on ozone was positive and significant at the 10 percent level (see equation (9), for example). In addition sulfur dioxide concentrations were positively and significantly related to the probability of chronic respiratory disease (CRD). This latter finding was consistent throughout

much of the analysis using the multi-year data set. Among the adults analyzed in the report, this was the one strong association to occur frequently. The lack of any significant findings with respect to ozone persisted across a number of sensitivity tests.

There was one exception to the negative findings regarding ozone and CRD. This occurred when the adult sample was divided into three groups consisting of those who had never smoked, those who were ex-smokers, and those who were current smokers. Among the never-smokers, ozone (measured by the multi-year average) was positively and significantly associated with CRD (Table 4-15, equations (23) and (26)). Among the never and former smokers, sulfur dioxide was positively and significantly associated with CRD.

In the analysis of CRD among children, there was but one positive and significant association--that concerned nitrogen dioxide in one model (equation (32), Table 4-16). As in the case of acute illness among children, very little sensitivity analysis was conducted because of the largely negative findings.

Brief attention was devoted in Volume I to chronic cardiovascular disease (CCD) (see Table 4-17). Explorations here found multi-year average ozone concentrations positively but never significantly associated with CCD. Nor were any such associations identified for other of the air pollutants considered. Confidence in these findings was bolstered by the fact that other risk factors identified in previous studies of CCD all had the expected sign and were highly significant in the analysis. These included age, race, smoking habits, weight, and education. In addition to

CCD, a variety of chronic diseases, neoplastic and otherwise, were examined for possible links to ozone and other air pollutants. No such associations were found. Once again, a caveat to this analysis is that only the most superficial tests were conducted. It is always possible that additional analyses might produce different results.

Volume II

Volume II of the Draft Final Report requires virtually no summary. Its primary purpose in to present the Health Interview Survey for 1979 and the two supplements, one on smoking and the other on residential mobility (which is actually pp. 110-111) of the HIS itself). In addition, Appendix B of Volume II contains the data documentation and cross-referencing. Appendix C (actually the third part of Volume II) does present research output from the RFF ozone project. However, this output is theoretical rather than empirical.

Specifically, Appendix C is a paper deriving the conceptually correct measure of the benefits of a pollution control program in a simple model of consumer behavior. In the model, pollution not only affects welfare by leading to illness but also enters individuals' utility functions directly. In addition, in the model individuals can protect themselves against illness by making "defensive" expenditures. (these might be purchases of air conditioners or filters, water purifiers, preventive health measures, and so on.) It was the purpose of this work to see how the presence of such defensive or averting expenditures might affect the valuation of health benefits, and how the correct valuation compared to the

out-of-pocket and opportunity costs associated with pollution-induced illness.

The results can be summarized as follows. First, the measure of an individual's willingness-to-pay for a pollution reduction contains only derivatives of the dose-response function, specifically those relating to the effect of pollution on health and defensive expenditures on health. Next, the conceptually correct measure will in general not only exceed the change in out-of-pocket and opportunity costs of illness but also the sum of these costs plus the change in averting or defensive expenditures. Finally, this conclusion holds for several different variations of the basic model in which workers are given paid sick leave, sickness affects the wage rate, and other changes as well.

Volume III

The third volume of work completed by RFF for OAQPS is more recent than the first two. It represents work undertaken since the April 1984 Public Peer Review Meeting and consists largely of additional analysis done in response to comments received at that meeting or elsewhere.

Chapter 2 is methodological. It discusses in some detail the kinds of problems that can arise when ordinary least squares techniques are used to estimate the likely effects of air pollution on health outcomes. On account of these problems--the most important of which is the frequent truncation or censoring of the range of health outcomes--alternative estimating techniques are reviewed. These include Tobit and Cragg-class models, truncated normal estimation, the sample selection model, Poisson

and geometrically distributed measures of health outcomes, and multinomial techniques.

The third chapter of Volume III is quite brief and is designed to answer a specific and important question: How would the analysis in Volume I have changed if the air pollution readings assigned to individuals had come not just from the single nearest monitor but from all monitors within some specified distance of the individuals' homes?

The answer suggested by the analysis in Chapter 3 is "Not very much at all." This conclusion follows from the simple correlation coefficients between the nearest monitor readings and the readings averaged over all monitors within ten and then twenty miles of the respondents' homes. For ozone the correlation coefficients are 0.97 and 0.93 for the ten and twenty mile averages, respectively. Thus, substituting either of the latter two for the nearest monitor readings would make virtually no difference in the results. The smallest of all the correlation coefficients thus calculated for all pollutants was 0.82.

Chapter 4 presents the results of some substantial reanalysis of the Volume I findings concerning ozone and acute respiratory disease. It differs from (and improves upon) the original work in several important respects. First, Poisson regression is used in place of ordinary least squares. For reasons discussed in Chapter 2 of Volume III, this is a more appropriate way to model acute health outcomes. Second, no individual was included in the analysis if the nearest air pollution monitor was more than ten miles from his home, as opposed to twenty in the original analysis. Third, the analysis of possible non-linearities is more sophisticated in

the later analysis. Finally, air pollution was measured not only by the reading at the nearest monitor during the two-week recall period but also by the average reading during that period of all monitors within ten and also twenty miles. Annual average readings were also employed in several of the models estimated.

The analysis in Chapter 4 supports the findings in Volume I. Specifically, ozone (however measured) is found to be positively and significantly associated with the number of restricted activity days due to respiratory disease during the two-week recall period. No effect is found for sulfates, the other air pollutant included in the Chapter 4 analysis. Of the other independent variables, race, income, the presence of a chronic illness, and temperature were also found to be associated in a statistically significant way with acute respiratory disease.

In other findings in Chapter 4, the possibility of interactive effects between ozone and sulfates and ozone and temperature are rejected. In addition, no evidence is uncovered supporting the idea of a "threshold" below which ozone concentrations are harmless. However, the analysis does support the finding that the dose-response relationship between ozone and acute respiratory disease is non-linear. That finding (see equation 4.5 in Table 4-4) suggests that the square root of the average daily maximum ozone concentration at the nearest monitor is a potentially important and significant determinant of acute respiratory disease.

Chapter 5 summarizes recent research on the construction of proxy measures for lifetime smoking profiles. The smoking controls used in both the statistical analyses in Volume I and in Chapter 4 of this volume were

typically either the current rate of daily cigarette consumption or dummy variables indicating whether the individual was a current, former, or never smoker. As noted at various points in the analysis, such variables are acceptable controls for assessing the possible linkages between current cigarette consumption and respiratory illness. However, because the relationship between cigarette smoking and respiratory illness is also thought to have lagged or cumulative dimensions, separate explanatory variables are required to effect controls for such relationships.

Given this requirement, and since no single value given in the data provides such information, we explored the detailed data available in the HIS Smoking Supplement in order to assess whether plausible proxies for lifetime cigarette consumption could be created. Using information on peak and current rates of consumption, age, age started smoking, and duration since quits, it was ascertained that various plausible controls for lifetime smoking could indeed be obtained from the HIS data. It turns out that the measures are created in a manner analogous to how capital stock values are obtained from investment and depreciation profiles in applied microeconomic research. Chapter 5 presents in considerable detail the procedures used for creating these lifetime cigarette consumption profiles.

Drawing on the results of Chapter 5, Chapter 6 proceeds in several directions to a more sophisticated analysis of the relationships between ambient air pollution and respiratory illness. Not only are the lifetime smoking profiles discussed in Chapter 5 used to control for longer-term or cumulative relationships between cigarette smoking and respiratory illness, but a new, and we believe particularly interesting, measure of respiratory

illness is used. That is, we consider in Chapter 6 the distinction between minor limitations in activity due to respiratory illness, i.e., those where the individual is not confined to bed, and major restrictions in activity due to respiratory illness, i.e., those where some bed confinement occurs. Moreover, we also consider in Chapter 6 the distinction used by the National Center for Health Statistics between inherently acute and inherently chronic respiratory illness.

To assess econometrically the relationships between the explanatory covariates and the new measures of respiratory illness, we use a multinomial logit estimator of the determinants of the various respiratory illness outcome probabilities. Like Chapter 4, it is found that nonlinear transformations of the ozone variable, as well as of the smoking controls, give superior results. Ozone is found to be positively related to the probability of all the respiratory illness outcomes, and the relationship is statistically significant for the minor or non-bedridden illnesses. The results also demonstrate that the additional effort involved in constructing the lifetime smoking profiles was worthwhile: in many of the specifications of the respiratory illness measures, both the current rate of consumption and the cumulative lifetime consumption were statistically important determinants of the probability of respiratory illness. In addition, the last section of Chapter 6 produces some calculations of the relative risks of various respiratory illness outcomes attributable to plausible hypothetical changes in ambient air pollution concentrations and current and lifetime cigarette consumption.

Chronic rather than acute illness is the focus of the seventh chapter of Volume III. In particular, the focus there is on the possible role of ozone, sulfates, and total suspended particulate matter in chronic respiratory disease (CRD). The analysis in Chapter 7 improves upon the analysis of CRD in Volume I in several ways. First, the sample of individuals used was restricted to respondents who has been living in their present location for ten years at the time of the 1979 HIS (compared with five years in the original work). Also, the more sophisticated measures of lifetime smoking described in Chapter 6 were used. Third, the sample of respondents was divided into two groups: one group was comprised of individuals who received the special "probe" questions in the 1979 HIS pertaining to respiratory disease; the other group consisted of individuals who received one of the five other disease probes. The sample was divided in this way because of evidence of differential response rates.

The results of this reanalysis of air pollution and CRD are ambiguous, in much the same way as were the findings in Volume I. For instance, when ozone was measured by the 1979 annual average daily maximum at the nearest monitor, it was positively and significantly related to CRD in the "probe" group. When averaged over more than one monitor in an area, the coefficient on ozone declined somewhat in significance, and it was by any reasonable standard indistinguishable from zero where the multiyear ozone data were used. . In regressions using respondents from the probe group, neither sulfates nor total suspended particulates were significantly associated with CRD, nor were any of the other independent variables--including the smoking measures. In the regressions run using

the non-probe group, ozone was never significantly associated with CRD. However, when the multi-year averaged air pollution data were used, particulates were positively and significantly associated with CRD. So, too, were both current and prior smoking habits, as well as income.

On the whole, then, reflecting on both the original work and the recent reanalysis, no consistent relationship between ozone (or any other air pollutants) and CRD has emerged in the RFF work. It is an area of continuing investigation, however.

Chapter 8 presents the results of a variety of sensitivity analyses of the Volume I research. That is, the results discussed in this chapter summarize analyses of the sensitivity of the Volume I empirical results to different assumptions about various data and model specification issues. Specifically, Chapter 8 treats the following topics. Discussed first is the question of proper specification of the precipitation measures. Our analysis suggests that the measures used in the models in both Volume I and in several of the chapters of Volume III are appropriate. Second, we assess the effects of sample size and selection on a set of the estimates presented in Volume I. It is found that the implications of varying sample size are largely manifested in the efficiency properties of the parameter estimates rather than in inferences about the central tendencies of the estimates themselves.

The third section of Chapter 8 presents a reestimation using Poisson regression of some of the models estimated by ordinary least squares in Volume I. The upshot of this exercise is that the alternative Poisson estimation techniques appear to be superior on statistical grounds.

However, the inferences about the relationships between ozone and respiratory health drawn from the corresponding models in Volume I are largely corroborated by the reanalysis. The analysis discussed in Section 8.4 concerns the question of structural aggregation. Specifically, this section examines whether structures of the relationships between explanatory variables and respiratory health outcomes differ for individuals with different cigarette smoking behaviors or with different chronic illness statuses. The results generally suggest that the hypothesis of structural homogeneity across the various smoking status or chronic illness categories be rejected.

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